We agree with Dr Berthelot that technical details, such as storage time of samples, should be included in APF studies. It is nevertheless difficult to evaluate retrospectively the effect of storage time or patient's age on APF positivity. For two of our patients, we assayed three serum samples collected over a period of four years and stored for one to five years. In each case, the initial APF was negative, but the following two were positive. However, it is difficult to conclude if this is a result of a shorter storage time, the patients growing older, or other variables. In our study, APF was most prevalent in the polyarticular onset, seropositive JRA group, which typically consists of older children, mostly teenagers. However, it is not clear if this is related to the patients' ages or, most likely, to their type of JRA, which closely resembles adult RA. We did not find any difference in APF occurrence between younger and older JRA patients with pauciarticular and seronegative polyarticular onset diseases.

These data underline the observation that APF testing can be influenced by many variables, especially in children, thus emphasising the need to define APF positivity in JRA on the basis of universal criteria.

GIDEON NESHER
THOMAS G OSBORN
Department of Internal Medicine
Doisy Hall, Saint Louis University Health Sciences Center, School of Medicine
St Louis, MO 63104, USA

TERRY L MOORE
Department of Internal Medicine and Pediatrics

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LETTERS TO THE EDITOR

Chronic calcifying pancreatitis in rheumatic diseases

We describe chronic calcifying pancreatitis (CCP) in two patients with rheumatic diseases.

A 61 year old female presented in May 1991 with acute pancreatitis caused by cholelithiasis; she underwent a cholecystectomy. Six months later, because of increased alkaline phosphatase concentrations an abdominal computed tomography (CT) scan was undertaken and revealed dilatation of bile and pancreatic ducts. This observation was confirmed by endoscopic retrograde cholangiopancreatography (ERCP) (fig 1); in addition, pancreatic tests were subnormal. However, the patient remained asymptomatic. In May 1992 she was evaluated in the rheumatology service where the diagnoses of systemic lupus erythematosus (SLE) and Sjögren's syndrome (SS) were made. Prednisone 5 mg/day was given for two months and chloroquine thereafter; diabetes was diagnosed in August 1992. In April 1993 the patient reported a mild epigastric pain and an abdominal CT scan revealed pancreatic calcification and a pseudocyst in the tail.

The second patient was a 51 year old female followed in the rheumatology service since 1978 with the diagnosis of scleroderma. She received as treatment indomethacin, metoclopramide, and ranitidine; she required the introduction of insulin in 1983, but never received steroids. In August 1992, after an episode of mechanical lumbar pain, calcifications were noted at the level of the pancreas on plain radiographs. Evaluation in the gastroenterology service revealed subnormal pancreatic tests, and pancreatic calcifications on abdominal CT scan (fig 2). ERCP revealed dilatations and stenoses of the main pancreatic duct, with normal bile ducts.

Figure 1 Endoscopic retrograde cholangiopancreatography showing mild dilatation of the common bile (arrowhead) and pancreatic (arrow) ducts.

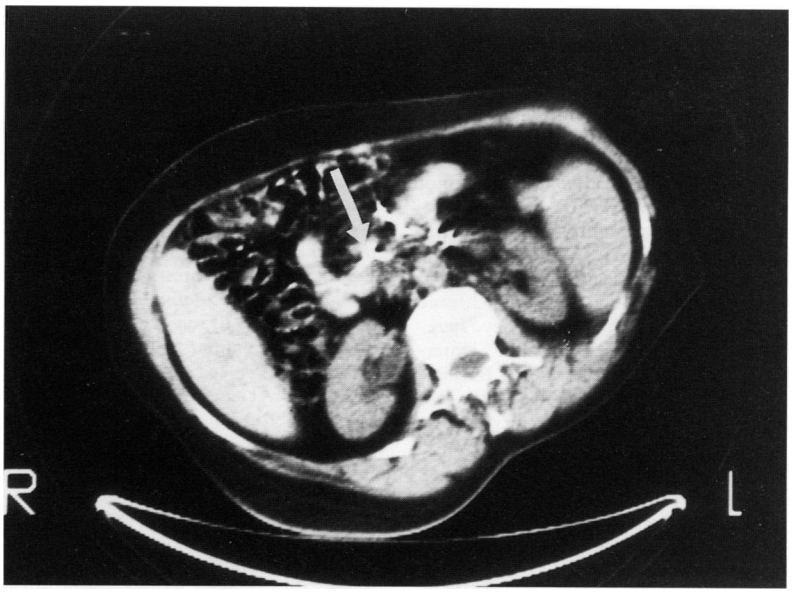

Figure 2 Abdominal computed tomography scan showing pancreatic calcifications (arrow) with global pancreatic atrophy.

Acute pancreatitis is the commonest clinical pancreatic manifestation in SLE.[1] Corticosteroids or immunosuppressive therapy are believed to be the causes, but immunological[2] and vasculitic[3] mechanisms are also important. Chronic pancreatitis in SLE has been reported only recently.[4]

Pancreatic problems have been reported rarely in SS, though there is evidence of subclinical insufficiency in SS,[5 6] ranging from 7% to 60%. Pancreatic tests in scleroderma have shown insufficiency in about 30% of patients but the nature of the disease is at best equivocal, as malabsorption may have other causes which cannot be distinguished easily by available tests; for example, hypoperistaltism with bacterial overgrowth or even reflux of duodenal contents into the pancreatic duct may affect the pancreatic exocrine function without necessarily indicating primary pancreatic involvement.

The pancreas has a potential for calcification in chronic pancreatitis, especially in alcohol or nutritionally-induced disease. The mechanism is not clear, but it seems that calcium may precipitate when the concentration of lithostatin, which maintains calcium solubility in the pancreatic juice, is reduced,[9] or when there is a decrease in volume flow with a high protein content of the pancreatic juice.[10] Theoretically, either or both mechanisms could be mediated by the pathogenetic processes of the collagen diseases.

Our first patient displayed several features that deserve remark: (a) the association of CCP with two conditions (SLE and SS) that independently predispose to pancreatitis; (b) onset of acute pancreatitis one year before the onset of SLE and SS; (c) mildness of SLE and SS; and (d), in common with our patient with scleroderma, a nearly asymptomatic course to calcification. It is possible that the modest dose of prednisone given for two

months, which unmasked the diabetes and prompted its discontinuation, could have had a pathogenetic effect, although this point is speculative. Presumably gall stones were the initial cause of the acute pancreatitis; however, we believe that they were not the cause of CCP, because a follow up CT scan failed to show them, and because gall stones are not a cause of chronic pancreatitis or CCP.[11]

In summary, we present two cases of CCP, one in a patient with SLE and SS, and the other in a patient with scleroderma, in which the usual causes of calcification—chronic alcohol intake, nutritional deficiency, hereditary pancreatitis, and chronic hypercalcaemia—have been excluded. Because there was no definitive aetiological factor, this could be either idiopathic and unrelated to the rheumatic disease, or a real association with these collagen diseases.

C HORTAS
Rheumatology Service, University Hospital 'Marqués de Valdecilla', Santander, Spain

G DE LAS HERAS
M J LÓPEZ-ARIAS
L MARTÍN
F PONS-ROMERO
Gastroenterology Service

Correspondence to: Dr C Hortas, Hotel San Millán, Peñacastillo-Camarreal, 15-B 39011 Santander, Spain.

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Small bowel telangiectasia in scleroderma

Scleroderma may involve the gastrointestinal (GI) tract in over 50% of patients, common problems being those of dysmotility, malabsorption and bacterial overgrowth.[1] Winterbauer documented gastric telangiectases in his case reviews in 1964,[2] and since then there have been sporadic reports of significant gastrointestinal bleeding from both gastric and colonic lesions.[3–9] We report the occurrence of telangiectasia of the small bowel in a patient with scleroderma and significant GI bleeding.

This 47 year old woman was diagnosed as having scleroderma when she presented with tightness, swelling of her fingers and Raynaud's phenomena. Her autoantibody profile, specifically anticentromere and anti-Scl-70, had always been negative. An upper GI endoscopy was performed when she developed symptoms of gastro-oesophageal reflux; this showed gastric antral telangiectases in a 'water melon' pattern. At that time her haemoglobin concentration was normal. Eighteen months later she was admitted as an emergency, having had melaena. Urgent repeat endoscopy demonstrated the previous lesions, but with no evidence of bleeding; subsequent colonoscopy was unremarkable. She continued to have episodes of blood loss and needed regular admissions to hospital for blood transfusions—approximately 30 units over six months. Coeliac axis and superior mesenteric angiography was performed and revealed no arteriovenous malformation.

Laparotomy and 'on-table' enteroscopy were carried out and revealed multiple telangiectases throughout the small intestine, the distal half being most densely affected. Three separate 4–7 cm lengths of the most severely involved ileum were resected. The patient still required frequent transfusions. However, as the relative contributions of the small bowel and gastric lesions to the overall blood loss were not clear a ^{99}Tc labelled red cell scan was undertaken. Its appearance was consistent with a source of bleeding in the terminal ileum. Because her gastric lesions might, at other times, be contributing more than was demonstrated, it was decided to treat these with heater probe ablation. Fortunately, her transfusion requirements have now reduced and she maintains a stable haemoglobin concentration with oral iron therapy alone.

Angiodysplasia may be responsible in up to 8% of cases of upper GI bleeding and possibly around 6% of bleeding of the lower GI tract.[10] Its symptomatic association with scleroderma seems rare, though when it does occur it is certainly significant. In this patient, small bowel lesions were not only shown to be present, but at one time appeared to be the main source of bleeding.

This finding is not surprising but appears not to have been described previously. The gastric telangiectasia is probably not without blame, but it is difficult to determine what proportion of blood loss has come from any particular site. Interestingly, this lady has never displayed cutaneous telangiectasia, unlike most of the cases previously reported. Should this lady's transfusion requirements again increase, it is proposed to try further ablation therapy, and then oestrogen therapy in an attempt to reduce small bowel blood loss.[10] We highlight this case in order to bring attention to the possibility of small bowel telangiectasia causing blood loss in patients with scleroderma.

G VAUTIER
E McDERMOTT
J E CARTY
B B SCOTT
Department of Medicine County Hospital, Lincoln LN2 5QY United Kingdom

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Correction

Anticardiolipin, anticentromere and antiScl antibodies in patients with systemic sclerosis and severe digital ischaemia

An author's error occurred in this paper by Dr Herrick and others (*Ann Rheum Dis* 1994; **53:** 540–2). The fifth sentence in the second paragraph on page 542 should have read: '... but a Spanish study ...' [not '... but another Japanese study ...'].

The authors wish to apologise to Dr Fonollosa and colleagues (Barcelona) for this mistake.